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Clinical letter

Reversible homonymous hemianopia secondary to occipital lobe seizures

R. Sawaya [a,*], W. Radwan [a], R. Alameddine [b], S. Hammoud [a]

[a] Department of Neurology, American University Medical Center, Beirut, Lebanon
[b] Department of Ophthalmology, American University Medical Center, Beirut, Lebanon

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1. Introduction

Occipital lobe infarcts, hemorrhages, or metastatic tumors can present with complex visual hallucinations, sensory or proprioceptive abnormalities, speech dysfunction, visual loss, and homonymous hemianopia (HH).[1–3] Treatment of occipital lobe seizures is by single or multiple antiepileptic drugs. We present a case of idiopathic occipital lobe epilepsy where the seizures were controlled by antiepileptic drugs, but the HH did not subside until treated with high dose intravenous (IV) steroids.

2. Case report

A 53-year-old man with uncontrolled hypertension, diabetes mellitus, and dyslipidemia presented with a 10-day history of episodes of complex visual hallucinations, distorted imagery, phosphenes and metamorphopsia. These episodes had gradually become more frequent and longer over this time. The episodes started and subsided spontaneously, lasting a few minutes, and occurring several times per day. They were associated with speech arrest, unresponsiveness, rotation of the head to the left, left gaze preference and tonic spasm of the left arm lasting for a few seconds. Physical examination on admission revealed a persistent left HH. Cranial nerve examination was normal, with normal eye movements and fundi. Speech was normal, but speech arrest was observed whilst the patient was experiencing the visual phenomena. Cognitive functions and memory were intact. No motor weakness or sensory dysfunctions could be elicited. Deep tendon reflexes were depressed. No meningeal or cerebellar signs were noted. Plantar responses were extensor bilaterally.

Vital signs were normal. Basic hematologic and chemistry blood studies were normal except for a fasting blood sugar of 210 mg%. MRI of the brain with gadolinum revealed high FLAIR signal intensity in the cortex of the right cerebral hemisphere with mild restriction on diffusion weighted images and no enhancement (Fig. 1c). No infarcts or tumors could be seen. MRA showed patent carotid, vertebral and cerebral arteries. EEG showed near continuous high frequency spike and sharp slow wave activity emanating from the right temporo-parieto-occipital region, which increased during hyperventilation and subsided immediately after intravenous diazepam 5 mg (Fig. 1b). The visual hallucinations resolved with the resolution of the seizure activity, but the hemianopsia persisted. Ophthalmologic examination was normal except for a complete HH confirmed by perimetry (Fig. 1a). CSF studies revealed normal cell count, an elevated protein (1.1 g/l), normal glucose and IgG level. The CSF gram stain was negative as were culture, acid fast and potassium hydroxide stains, brucella and HIV, HTLV I and II serology, PCR was negative for HSV, VZV, EBV, CMV and JC viruses. CT chest, abdomen and pelvis with contrast were normal. The patient was treated with phenytoin, valproic acid and levitracetam. The visual hallucinations, head tilting, speech arrest and tonic arm spasm subsided, but the HH persisted clinically and by perimetry. Follow-up EEG showed resolution of the epileptic activity, and repeat MRI a marked

* Corresponding author at: Clinical Neurophysiology Department, American University Medical Center, P.O. Box: 113 6044/C 27, Beirut, Lebanon.
Fax: +961 1 354305/365612.
E-mail address: rs01@aub.edu.lb (R. Sawaya).

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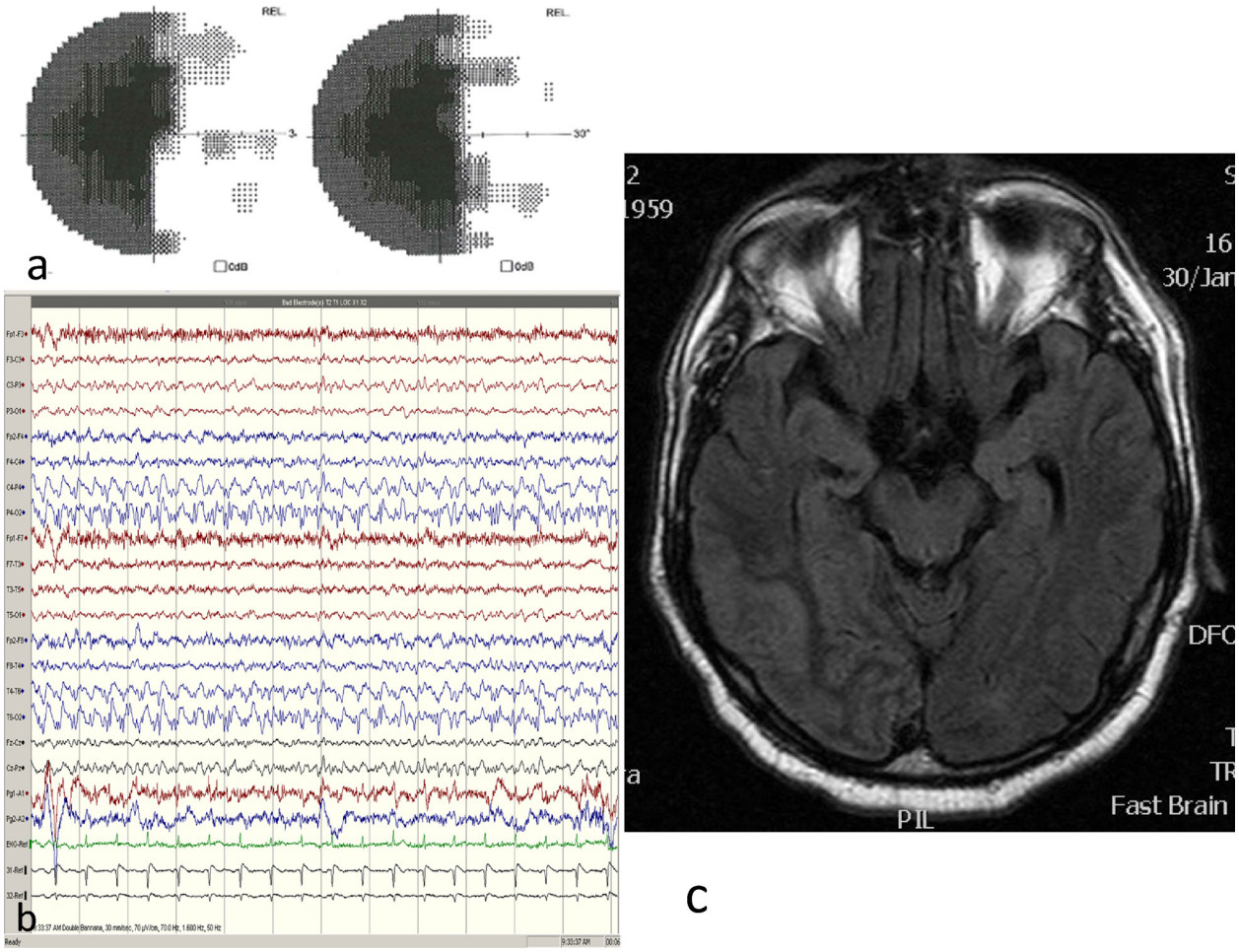

Fig. 1. (a) Visual field perimetry showing complete right homonymous hemianopia. (b) Epoch of a 21-electrode EEG showing continuous epileptic activity over the right occipital electrodes. (c) Axial MRI of the brain showing high FLAIR signal intensity in the right cerebral hemisphere.

decrease in the right parieto-occipital hyperintensity and edema. The patient was treated with 1 g methylprednisolone intravenously resulting in the resolution of the HH. The patient was discharged on a tapering dose of oral steroids and antiepileptic drug therapy.

3. Discussion

HH can be complete or incomplete and is caused by a lesion affecting the contralateral retrochiasmal visual pathway. Lesions of the optic radiation in the temporal lobe can produce an incomplete HH with aphasia, complex partial seizures, memory deficits and hallucinations. Lesions of the optic radiations in the parietal lobe may also produce also an incomplete HH with hemineglect, sensory deficits or Gerstmann syndrome.[4]

Visual field defects secondary to occipital lobe seizures may be quadrantic, hemianopic, or total. The etiology of the seizures can be vascular, tumoral, idiopathic or cryptogenic.[1–3] Focal seizures and their manifestations usually respond to antiepileptic therapy. Associated hemianopia may resolve spontaneously, following antiepileptic therapy, or may persist.[3]

Our patient, a middle aged man with multiple risk factors for cerebral strokes, presented with a clinical picture suggestive of right posterior cerebral artery infarction complicated by epileptic seizures. However, the diagnosis of stroke could not be confirmed by MRI or MRA of the brain and the symptoms eventually resolved without any treatment for a cerebrovascular insult. The patient most probably did not have a stroke. The patient's HH which is likely to have been part of the symptomatology of his occipital lobe seizures did not subside with the resolution of the other seizure symptoms, or the epileptic activity on the EEG, upon administration of intravenous benzodiazepine or maintenance antiepileptic treatment. The HH subsided only after the first high-dose intravenous steroid therapy. This suggests that the HH was the result of edema of the temporo-parieto-occipital region caused by focal status epilepticus or frequent focal seizures. There is no clear explanation why this patient developed a focal status epilepticus without any evidence for a stroke, hemorrhage, subdural hematoma, metabolic derangement, infection, malignancy, meningitis or encephalitis.

We suspect that the pathophysiology of the HH is inflammatory rather related to a derangement in ion channel function, as it resolved with steroids and not antiepileptic treatment. We classified the epilepsy as idiopathic or cryptogenic, as no cause for the seizures was found despite extensive investigation.

In conclusion, occipital lobe epilepsy can be cryptogenic, and present with complex visual hallucinations and HH. Seizures may be controlled with multiple antiepileptic drugs. Persistent HH should be treated with high-dose intravenous steroids to avoid permanent visual loss.

Conflict of interest

None declared.

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